Table 6-11. Peak groundwater concentrations for WAG 4.

Contaminant	Modeled Decay Product[a]	Peak Concentration[b] (mg/L or pCi/L)	Time (yr) at Peak Concentration
Ac-228 (Th-228)[c]		0.00E+00	n/a[d]
Ag-108m		0.00E+00	n/a[d]
Am-241 (Np-237)[c]		3.47E-05	2.64E+03
	U-233	3.31E-07	
	Th-229	2.31E-09	
Ba-133		0.00E+00	n/a[d]
Bi-212 (Pb-208)[c]		n/a[e]	n/a[e]
Bi-214 (Pb-210)[c]		0.00E+00	n/a[d]
Cs-137		0.00E+00	n/a[d]
Eu-152		4.79E-03	4.11E+01
Pb-212 (Pb-208)[e]		n/a[c]	n/a[e]
Pu-238 (U-234)[c]		2.56E-06	4.68E+03
	Th-230	5.43E-08	
	Ra-226	6.44E-07	
	Pb-210	3.24E-08	
Pu-239/240		1.06E-02	1.70E+04
	U-235	8.32E-07	
	Pa-231	1.01E-07	
	Ac-227	1.16E-08	
Ra-226		8.49E-09	2.20E+04
	Pb-210	8.61E-09	
Tl-208 (Pb-208)[e]		n/a[e]	n/a[e]
U-234		3.54E+00	1.35E+03
	Th-230	2.50E-02	
	Ra-226	6.20E-03	
	Pb-210	5.94E-03	
U-235		2.56E-01	1.35E+03
	Pa-231	5.08E-03	
	Ac-227	5.70E-04	
U-238		3.91E+00	1.35E+03
	U-234	1.49E-02	
	Th-230	5.38E-05	

Table 6-11. (continued).

Contaminant	Modeled Decay Product[a]	Peak Concentration[b] (mg/L or pCi/L)	Time (yr) at Peak Concentration
	Ra-226	9.20E-06	
	Pb-210	8.61E-06	
Zr-95 (Mo-95)[f]		n/a[f]	n/a[f]
1,1,1-Trichloroethane		6.22E-08	1.20E+02
Arsenic		4.40E-02	6.96E+02
Benzo(a)anthracene		1.23E-08	2.88E+05
Benzo(b)fluoranthene		1.85E-09	8.92E+05
Benzo(g,h,i)perylene		1.83E-09	1.15E+06
Chlorodifluoromethane		1.74E-04	7.79E+01
Di-n-butylphthalate		2.98E-06	2.22E+04
Lead		1.33E-03	5.02E+04
Mercury		1.03E-02	2.18E+04
Phenanthrene		3.83E-08	2.42E+04
Phenol		7.10E-05	5.91E+01
Tetrachloroethene		1.05E-07	2.29E+02
TPH-diesel[g]		3.74E+00	4.27E+02
TPH-gasoline		6.89E+00	3.30E+02
TPH-heating		7.95E+00	4.64E+02

a. Some radionuclide COCs decay to significant daughter products; the daughter product ingrowth is included here. For this analysis, daughter products are assumed to travel at the same rate as the parent.

b. The groundwater concentrations reported in this table represent the maximum predicted in a network of ten receptor aquifer wells located in a line perpendicular to the flow direction immediately downgradient of the reference site (CFA-04).

c. Radionuclide contaminants that have short half-life relative to the vadose zone transit time were modeled as their first radioactive decay product. These include Ac-228, Am-241, Bi-214, and Pu-238 which were modeled as Th-228, Np-237, Pb-210, and U-234, respectively.

d. Some radioactive contaminants decay to stable products before reaching any receptor well locations.

e. Some radionuclides with very short half-life (<1.0 yr) that have no significant radioactive decay products were modeled as stable decay products. Bi-212, Pb-212, Tl-208 soil inventories were converted to stable lead which was added to the total lead inventory (see results for lead).

f. Zr-95 is also very short-lived with no significant radioactive decay products; the inventory of Zr-95 was converted to stable Mo-95, which was found to be an insignificant soil inventory relative to the molybdenum MCL.

g. TPH = total petroleum hydrocarbon

Assumptions

The contaminant concentrations shown in Tables 6-10 and 6-11 are expected to overestimate the true aquifer concentrations that will be produced by infiltration of contaminants at WAG 4. Because of the complicated flow and transport of contaminants in groundwater, the uncertainty about potential contaminant concentrations associated with the groundwater pathway exposure routes is greater than the uncertainty associated with any other exposure pathway in this BRA. To compensate for this relatively large uncertainty, assumptions are used throughout the groundwater pathway analysis that will cause calculated COPC concentrations to be higher than would be expected. Some of the conservative assumptions that are used in the GWSCREEN analysis are as follows:

- For the purposes of groundwater modeling, infiltration of precipitation at WAG 4 is assumed to be spatially uniform. Increase in the value of this parameter at one site or another due to accumulation of runoff from other areas and, conversely, decrease in the value due to drainage of runoff are not considered. The INEEL Track 2 default value of 0.10 cm/yr is considered an acceptable value for this parameter.

- For the purposes of this modeling, the unsaturated zone is defined as the zone between the base of the source volume and the top of the aquifer. The unsaturated zone is assumed to be homogeneous, isotropic, porous media with constant, unidirectional flow in the vertical (downward) direction. Due to the complexities and inherent uncertainties in modeling unsaturated flow and transport, a simplified plug-flow model is used that does not account for dispersion in the unsaturated zone. In the plug flow model, non-sorbing contaminants move with the vertical velocity of the water. This velocity is calculated assuming the unit hydraulic gradient (gravity drainage) condition.

  For most contaminants, the plug flow model is a conservative assumption since the peak flux to the aquifer exceeds the peak flux when dispersion is considered. Although plug flow leads to higher predicted contaminant concentrations in the aquifer, predictions of contaminant travel time to the aquifer are lower when vadose zone dispersion is included. Recent versions of GWSCREEN incorporate vertical dispersion in the unsaturated zone; however, values for the unsaturated zone vertical dispersivity are currently gross estimates only.

- In the unsaturated zone, water and contaminants are assumed to flow through basalt sequences very quickly relative to flow through sediment. As a result, the accepted Track 2 method is to ignore the vertical thickness of basalt in the vadose zone. For modeling purposes, unsaturated flow is assumed to occur through surficial and interbed sediments only.

  The accepted Track 2 method for estimating the vadose zone thickness, devoid of basalt, is to sum the known surficial sediment thickness and interbed thicknesses between the base of the source and the top of the aquifer or, when little subsurface data are available, to use one-tenth the overall depth to the aquifer. For the WAG 4 groundwater modeling, available lithological data from well logs were analyzed to provide site-specific cumulative interbed thicknesses.

  For non-decaying contaminants, the only effect variations in the total unsaturated zone sediment thickness have are on the unsaturated transit time and, hence, contaminant arrival times at the receptor. All of the non-decaying contaminant will eventually reach the aquifer

regardless of the unsaturated sediment thickness. The sensitivity of groundwater modeling predictions to changes in this parameter are investigated in Appendix F.

- For radiological contaminants, the mass of contaminant reaching the aquifer and, hence, aquifer concentrations at the receptor are affected by changes in the unsaturated zone sediment thickness. Decay of the radiological contaminant in the unsaturated zone will be enhanced by greater sediment thicknesses. Large adsorption coefficients for some radiological contaminants will further enhance this vadose zone decay.

- All COPC mass contained in surface soils at WAG 4 is assumed to contribute to groundwater contamination. No credit is taken in the modeling for loss of COPC mass caused by mechanisms such as wind erosion, surface water erosion, contaminant uptake into plants, biodegradation, etc. The only contaminant loss mechanism that is considered in the groundwater pathway evaluation is radioactive decay.

Two other assumptions included in the groundwater analysis, but not limited to the GWSCREEN modeling, are as follows:

- The groundwater receptor is assumed to take all drinking water for 30 years from one of ten receptor well locations along the downgradient edge (with respect to groundwater flow) of WAG 4.

- All contaminants are assumed to be uniformly distributed within the groundwater modeling source volume.

Model Selection

The groundwater modeling performed for the WAG 4 RI/FS is based on the solution algorithms of the semi-analytical model GWSCREEN (Rood 1994). GWSCREEN is used widely at the INEEL for assessment of groundwater pathway impacts from surface or buried contamination. The algorithms of one of the latest versions of the code, version 2.4a, are incorporated in a pre- and post-processing user-interface called GWMENU. This interface allows integration of input and output from the model for easier computation of cumulative impacts of more than one site at a common receptor.

Although formal documentation for GWMENU is not available, the interface's similarity to GWSCREEN version 2.4a is confirmed as part of the groundwater modeling sensitivity analysis included in Appendix F. Runs were made using the GWMENU program and compared with runs by GWSCREEN version 2.4a using the same input parameters.

The GWSCREEN family of codes was designed to perform groundwater pathway screening calculations for the Track 1 and 2 process. It is an appropriate model to use when site characterization data are lacking and little would be gained by the use of more complex models. Other more sophisticated models were reviewed but were not selected to perform groundwater pathway calculations because:

- The probability of potential risks exceeding acceptable criteria was low and did not warrant the use of a more sophisticated model at this time in the process,

- Additional field characterization data would likely need to be obtained to fully utilize the capabilities of a more complex model, and

- The results of this analysis will indicate what operable units, if any, require further attention; the use of a more complex model at such time may be justified.

Source Areas

Source areas were modeled individually instead of modeling a single WAG 4 composite site. Each release site within WAG 4 was represented by rectangular areas that were oriented parallel to north-south or east-west lines. Dimensions used in the model, as described in Table 6-7, are modified from actual site dimensions to account for irregular geometry and source orientation. The total volumes of each modeled source, however, were made to approximate the actual estimated contaminated volumes (see Table 6-7). A total of 13 sites, retained following the supplemental contaminant screening performed in this report (Section 6.2..2.2) were modeled.

Some of the retained sites required several rectangular areas (elements) to represent the source. These include CFA-05, CFA-07, CFA-17. CFA-05, the motor pool pond, includes the pond as well as a ditch (two elements); CFA-07 includes two french drains (two elements); CFA-17 includes a rectangular fire training pad and a leach pit (two elements). CFA-08 contains two elements also; one representing the sewage treatment plant, the other represents the drain field.

In addition, the modeling included 22 aboveground and underground fuel storage tanks identified in the facility analysis portion of the OU 4-13 Work Plan. Simplifying assumptions were used to incorporate these tanks for which little data are available. These include assuming only one tank volume of fuel product leaked from each tank into the subsurface during the life of the tank. This assumption is reasonably conservative because operators of the tank systems would have probably noticed larger leaks.

Each retained site was located according to its physical geographic location within the CFA facility. Two sites are located outside of CFA; although CFA-17 and CFA-47 are retained in the OU 4-13 effort, they are located on Lincoln Avenue approximately 6 km (4 mi) north of CFA. Table 6-7 presents the Universal Transverse Meridian (UTM) north and east coordinates for each site.

A receptor grid was overlain on the source areas such that contributions to individual contaminant groundwater concentrations from all retained sites could be calculated at each receptor node. CFA-04 served as the most downgradient site, with respect to groundwater flow, and as such served as the model reference site. With the exception of CFA-12, CFA-04 is also the western-most site that was modeled. Contaminant groundwater concentrations were determined for each of ten receptor locations spread across an east-west line at the downgradient edge of CFA-04 that extends from 200 m (658 ft) west of the center of CFA-04 to 1200 m (3,947 ft) east of CFA-04.

A common receptor grid allows groundwater concentrations of contaminants common to two or more sites to be summed for determining cumulative impacts. The concentrations reported in Tables 6-10 and 6-11 are these cumulative groundwater concentrations. More detail on the design of the groundwater model including the receptor network is provided with the sensitivity analysis and output files in Appendix F.

The spacing between receptors varies; the western most receptor is 200 m (658 ft) from the center of CFA-04, the next five receptor locations are spaced 100 m (329 ft) apart, and the final four receptors to the east spaced at 200 m (658 ft). This was done because the majority of sites modeled for the groundwater pathway are located within 500 m (1,645 ft) east of CFA-04. The distance from the center of CFA-04 to the center of each area source was computed (listed in columns 4 and 5 of Table 6-7) which provided a means to relate the receptor grid to all source areas in the model domain. Additionally, a

receptor well was located at the downgradient edge of each source. The results for the source edge are not reported in this section but can be found in the appendix containing model output (Appendix F).

Each source area was modeled as a surficial or buried source as described in the GWSCREEN user's manual (Rood 1994). These sources are modeled in GWSCREEN using the assumption of homogeneously mixed contamination within the source volume and with natural infiltration of precipitation as the only downward driver for contaminant migration. Steady state infiltration under unit hydraulic gradient conditions are assumed. Contaminants area assumed to be in solid and aqueous phase equilibrium and equilibrium concentrations are described by the linear sorption coefficient, K_d. Leaching from the source volume to the underlying strata is assumed to be a first-order process where the fraction leached is constant and the mass flux is proportional to the amount of contaminant present in the source volume. Radioactive decay is considered but all other loss mechanisms, including biodegradation of petroleum products, are ignored.

Unsaturated Zone

The Track 1 and 2 groundwater modeling process typically only considers water travel time through sedimentary interbeds and assumes the transit time through fractured basalt is relatively instantaneous. The Large Scale Infiltration Test, performed at the INEEL in 1995, came to a similar conclusion that transit time in the unsaturated zone is controlled by the hydraulic properties of the sedimentary interbeds and not the fractured basalt. This approach has been incorporated into this analysis. Interbed thickness is known to vary across CFA. Site-specific well logs have been used to delineate the interbed thickness below the retained sites. The advantage to the conceptual model employed in this analysis is that each source area may be assigned an interbed thickness that coincides with the actual estimated interbed thickness underlying the source. Using a single composite source does not allow for this refinement. The sensitivity of predicted groundwater concentrations to changes in the unsaturated zone sediment thickness are analyzed in Appendix F.

The unsaturated zone in GWSCREEN is modeled using a plug flow model. Dispersion and diffusion are ignored in the vadose zone; only radioactive decay is allowed to reduce contaminant concentrations in the leachate. Unit gradient conditions are assumed throughout the unsaturated zone. Contaminant travel times are governed by the water infiltration rate, contaminant-specific sorptive properties, and the hydraulic properties of the interbeds.

Saturated Zone

The saturated zone (Snake River Plain Aquifer) is modeled as a homogeneous isotropic aquifer of infinite lateral extent and finite thickness. No sources or sinks are considered and a steady state uniform flow field is assumed. Contaminants enter the aquifer in an area defined by the length and width of the source area and disperse both horizontally and vertically as they are transported downgradient. Equilibrium sorption reactions described by the K_d value are included. Contaminant concentrations are evaluated by averaging the concentration in the first 15 m (49 ft) of the aquifer (measured from the water table, the top surface of the aquifer). The 15 m (49 ft) averaging concentration depth was chosen based on the default Track 2 length of well screen (DOE/ID 1994). Contaminants are allowed to disperse completely in this effective aquifer thickness as they move downgradient.

Groundwater flow direction underneath CFA is approximately south to southwest. To simplify the GWSCREEN calculations, the flow direction is assumed to be directly south. This assumption may be nonconservative for some sites, overly conservative for others, but the overall effect is expected to be minimal. Gradients in the SRPA underneath CFA are relatively shallow and the actual flow direction is not precisely known. For sites with significantly different length and width dimensions, groundwater

flow direction will influence the estimated maximum concentration. Sources such as CFA-05 may generate higher groundwater concentrations when the groundwater flow is oriented in a more western direction. Groundwater concentration results were calculated primarily for groundwater flow in the north-south direction because:

- The estimated flow path is nearer to the north-south direction compared to east-west.
- Elongated source areas such as CFA-05 are actually more complex, containing several points at which source orientation changes with respect to groundwater flow.
- Groundwater flow orientation has little impact on maximum concentrations predicted for sources with approximately equal length and width.
- A change in groundwater flow direction will increase predicted concentrations from some source areas while decreasing concentrations from others.

To quantify the uncertainty associated with the groundwater flow direction and to provide the sensitivity of predicted groundwater concentrations to changes in the flow direction, the groundwater modeling sensitivity analysis in Appendix F includes analysis of flow direction. The model was rerun with different flow directions and the results compared with the base case (flow directly south) presented here.

Groundwater Transport Parameters

In this section, the input parameter values for the GWSCREEN model for each retained, modeled site source element are described. Parameter values are provided for the source, unsaturated, and saturated zone. Contaminant sorption coefficients values (K_d) used in this analysis and listed in Table 6-9 are the same as those used in the human health risk assessment and are shown by contaminant in Appendix F.

Source Area Parameters

Three input parameters shown in Table 6-7 (length of source parallel to groundwater flow, width of source perpendicular to groundwater flow, and thickness of source) are based on a variety of dimensional estimating methods. These include known estimates based on assumed contaminant release volumes, soil porosity, and depth to basalt; known extents of soil contamination, where available; and estimates based on the square root of a known surface contaminated area. Thickness of contamination was based on sampling results; however, vertical extent of contamination for the groundwater pathway includes an estimated depth to which contamination is suspected to have leached. Retained site source areas were oriented such that their sides were parallel to north-south or east-west trending lines. Tanks were oriented such that their longest dimension is parallel to the flow direction; extent of contamination was based on one tank volume released, residual soil porosity, and depth to basalt. Other tank input parameters are shown in Table 6-8.

Surface soil hydraulic properties were obtained from the GWSCREEN user's manual (Rood 1994). Default Track 1 and 2 moisture contents and an annual infiltration rate of 10 cm/y were used as input. For contaminated source zones, their respective infiltration rates provide the driving force for the transport of contaminant from beneath these surface features, through the remaining vadose zone, and into the aquifer. These infiltration rates are assumed to result from natural sources only. Natural sources of

infiltrating water include precipitation as either rain that falls directly on to the contaminated zone surface or snowmelt from snow that falls directly on the surface feature.

Natural infiltrating water can also include rainfall or snowmelt from other areas of the site that may flow as runoff and collect on the surface of contaminated zones. The natural background infiltration rate for these sites is a function of precipitation, evapotranspiration, and other factors. The widely accepted infiltration rate for the INEEL is taken from Track 2 guidance (DOE 1994) as 0.1 m/yr. This rate is assumed for all contaminated source zones modeled for the groundwater pathway. Potential runoff from other parts of the facility that may collect above contaminated zones is ignored in this analysis.

Unsaturated Zone Parameters

Interbed thickness is known to vary considerably across the INEEL. Well logs have been used to delineate thickness at different locations within WAG 4. As noted On Table 6-5, data available for associated delineation of the interbeds is limited. Isopleth contours of interbed thickness were generated using a Kriging interpolation routine that is part of the Surfer® software package (Golden Software Inc. 1996). All source areas are underlain by sedimentary interbeds of varying thickness. The sum thickness of the interbeds beneath a contaminated site and above the top of the aquifer is referred to here as the cumulative vadose zone interbed thickness (CVZIT). The CVZIT values for CFA area aquifer wells. used to prepare the kriged contours, are listed in Table 6-5. CVZIT values estimated for each modeled site are listed in Table 6-7.

The effects of varying the CVZIT parameter are analyzed in the sensitivity analysis of Appendix F. The input parameter values and modeled results presented in this section serve as the base case for the Appendix F analysis. The sensitivity analysis includes examination of effects of minimum and maximum unsaturated zone sediment thicknesses for each modeled site. It also includes the effects of discontinuous interbeds at WAG 4 and in areas downgradient of WAG 4.

Saturated Zone Parameters

Saturated zone parameters were obtained from the Track 1 and 2 guidance manuals (DOE 1992 and 1994) and are listed in Table 6-6. Values for effective porosity, dispersivity, and well screen thickness were obtained from the Track 1 and 2 guidance manuals. A pore velocity (average linear velocity) for the Snake River Plain Aquifer near CFA of 570 m/y is a default Track 2 parameter. The effective aquifer thickness was assigned a default value of 76 m.

The aquifer model used in this analysis included dispersion in three directions. Therefore, a vertical dispersivity value was required. Typically, the vertical dispersivity is assumed to be the same as the transverse dispersivity. In order to provide some conservatism to the calculation, vertical dispersivity was assigned a value 10 times less than the transverse dispersivity 0.4 m (1.3 ft). Contaminants were allowed to disperse into the effective thickness of the aquifer (76 m). Output concentrations were based on the average aquifer concentration in the first 15 m (49 ft) of aquifer (the well screen thickness) measured from the surface. Near the source, little vertical mixing occurs and calculated concentrations are similar to those calculated using a vertically averaged model and 15 m (49 ft) mixing thickness. Note, this is the same approach used in the Track 1 and 2 process. Farther away from the source, the contaminant plume disperses beyond the 15 m (49 ft) well screen depth. Therefore, contaminant concentrations are lower compared to those calculated assuming a constant 15 m (49 ft) mixing thickness.

Maximum concentrations in the aquifer were calculated for each contaminant; both the peak concentrations, occurring at any time, and concentrations occurring during 100–130 years from present

were obtained. The values presented in Table 6-10 and Table 6-11 are the highest 30-yr average concentrations of the set of ten receptor locations. Contaminant concentrations at each receptor node were calculated separately for each source and then summed across all sources. Therefore, concentrations for a specific contaminant at each receptor node represents contributions from all sources considered in the assessment.

Appendix F contains the GWSCREEN output files for the peak concentration simulations prepared for each COPC. Table 6-10 and 6-11 summarize the predicted groundwater concentrations.

It should be noted that short-lived radionuclides with significant (long-lived) decay products and high sorption coefficients were modeled as their first long-lived daughter product. This assumes the parent nuclide inventory will transform to the daughter product while still bound in the vadose zone. Am-241 and Pu-238 are two such parent radionuclide contaminants at WAG 4; they were modeled with GWSCREEN as Np-237 and U-234, respectively. Additionally, Ac-228 and Bi-214 were modeled as Th-228 and Pb-210, respectively.

Other short-lived radionuclide contaminants with no significant long-lived daughter products were modeled as their final stable decay product. Bi-212, Pb-212, and Tl-208 were all converted from curies of each to milligrams of stable Pb-208. These inventories of Pb-208 were then added to the total lead inventory at this site (CFA-05). The inventory of Zr-95, present at CFA-13, was converted from curies of Zr-95 (6E-06 Ci) to milligrams of stable Mo-95, which has a MCL of 0.18 mg/L. The resulting inventory of Mo-95 (2E-25 mg) is too insignificant to warrant further groundwater modeling.

Finally, the individual BTEX (benzene, toluene, ethylbenzene, and xylene) compounds, typically risk-drivers for petroleum contaminated sites, were not modeled separately for petroleum contamination sites at WAG 4. The petroleum release sites were modeled assuming the release of one-tank volume of petroleum product. The relative concentrations of BTEX compounds in petroleum products vary considerably and are a function of the proprietary mix of chemicals used by petroleum vendors. As a result, the petroleum sites were modeled as TPH of either gasoline, diesel, or heating oil.

6.3.3.3.1 Groundwater Ingestion—The groundwater pathway is evaluated on a WAG-wide basis. As with the soil and air pathways, groundwater pathway risks and HQs are calculated for the 100 year residential scenario.

6.3.3.3.2 Dermal Absorption From Groundwater—Exposures to COPCs through dermal absorption of groundwater are controlled by the chemical-specific permeability coefficient of water through skin (K_p^w). According to EPA guidance (EPA 1992c), if the permeability coefficient for a given COPC is less than 0.1 cm/hour, then the dermal absorption from groundwater exposure route results in potential risks that are less than potential risks via the groundwater ingestion exposure route for that COPC. In the BRA, the default permeability coefficient used for inorganic COPCs is 1E-03 cm/hour, and the permeability coefficients for organic COPCs are estimated using the following equation:

$$\text{Log } K_p^w = -2.72 + 0.71 \text{ Log } K_{ow} - 0.0061 \text{ MW} \qquad (6\text{-}13)$$

where

Kow = octanol/water partition coefficient (unitless)

MW = molecular weight (g/mol).

Permeability coefficients for WAG 4 COPCs are shown in Table D-14. Because many of the organics have permeability coefficients that are greater than the screening level of 0.1 cm/hr, the dermal absorption from the groundwater exposure route is quantitatively evaluated in the BRA. Contaminant intakes for this exposure route are calculated using the equations shown in Section 6.3.4.

6.3.3.3.3 Inhalation of Groundwater VOCs. Exposures caused by the inhalation of water vapors from indoor water use are calculated based on experimental data derived from a study of household water contaminants (Andelman 1990). This study derived a volatilization constant that defines the relationship between the concentration of a contaminant in household water and the average concentration of the volatilized contaminant in air. All uses of household water were considered (e.g., showering, laundering, and dish washing), and certain reasonable assumptions were made in deriving a volatilization fraction. For example, the study included assumptions about water usage for a family of four, the volume of the dwelling, and the air exchange rate. Furthermore, the study assumed that the average transfer efficiency weighted by the type of water use is 50% (i.e., half of the concentration of each chemical in water will be transferred into air by all types of water uses).

Indoor water use analysis, a central tendency value [6.50E–02 mg/m^3 air per mg/L water (Andelman 1990)] for the volatilization fraction of a COPC is used in the BRA to develop estimates of COPC airborne concentrations. The airborne concentrations are calculated by multiplying the central tendency value by the COPC groundwater concentrations shown in Table D-45. These concentrations are then used to develop contaminant intake estimates using the equations shown in Section 6.3.4. The estimates of COPC airborne concentrations from indoor water use calculated for the BRA are shown in Table D-22.

6.3.4 Estimation of Contaminant Intakes

The general equation that is used to calculate intakes for most of the BRA exposure routes is as follows (EPA 1989a):

$$Intake = \frac{C \times IR \times EF \times ED}{BW \times AT} \qquad (6\text{-}14)$$

where

Intake = contaminant intake (mg/kg-day)

C = concentration of a given contaminant in a contaminated medium (soil, air, water, etc.) (mg/kg, mg/m^3, mg/L, etc.)

IR = ingestion rate of the contaminated medium (mg/day, m^3/day, L/day, etc.)

EF = exposure frequency (day/year)

ED = exposure duration (year)

BW = body weight (kg)

AT = averaging time (day).

The above equation applies to all exposure routes except exposure to external radiation. For the external radiation exposure route, intakes are calculated using the following general equation:

$$\text{Intake} = C \times ET \times EF \times ED \times CF \quad (6\text{-}15)$$

where

Intake = radiation intake (pCi-year/g)

C = radionuclide concentration in soil (pCi/g)

ET = exposure time (hour/day)

EF = exposure frequency (day/year)

ED = exposure duration (year)

CF = conversion factor (1.14E–04 year/hour).

The exposure assumptions used to calculate intake factors for the occupational and residential exposure scenarios are summarized in Table D-23. Exposure pathway-specific intake factor equations are presented in Tables D-24 through D-35. Tables D-36 through D-39 present the calculated intake factors for the current and future occupational exposure scenario. Tables D-40 and D-41 present the calculated intake factors for the future residential scenario.

6.4 Toxicity Assessment

This section provides the toxicity constants that will be used for risk characterization purposes and summarizes toxicological information for the WAG 4 radioactive and nonradioactive COPCs. For this assessment, and consistent with *EPA's RAGS* (EPA 1989a), the toxicity information is summarized for two broad categories of potential effects: noncarcinogenic and carcinogenic effects. These two categories are selected because of the slightly differing methodologies for estimating potential health risks associated with exposures to carcinogens and noncarcinogens.

The toxicity constants used in the BRA are obtained from several sources. The primary source of information is the EPA's Integrated Risk Information System (IRIS). IRIS contains only those toxicity constants that have been verified by EPA's Reference Dose or Carcinogen Risk Assessment Verification Endeavor Work Groups. The IRIS database is updated monthly and supersedes all other sources of toxicity information. If the necessary data are not available in IRIS, EPA's HEAST (EPA 1995a) are used. The toxicity constant tables are published annually and updated approximately twice per year. HEAST contains a comprehensive listing of provisional risk assessment information that has been reviewed and accepted by individual EPA program offices, but has not had enough review to be recognized as high-quality, agency-wide information (EPA 1995a).

6.4.1 Toxicity Assessment for Carcinogenic Effects

Potential carcinogenic risks are expressed as an estimated probability that an individual might develop cancer from lifetime exposure. This probability is based on projected intakes and chemical-specific dose-response data called cancer SFs. Cancer SFs and the estimated daily intake of a compound, averaged over a lifetime of exposure, are used to estimate the incremental risk that an

individual exposed to that compound may develop cancer. This estimate is derived using the following equation:

$$\text{Risk} = \text{Intake} \times \text{SF} \qquad (6\text{-}16)$$

where

Risk = Carcinogenic risk (unitless)

Intake = Chemical intake (mg/kg-day or pCi)

SF = Slope factor ($[\text{mg/kg-day}]^{-1}$ or $[\text{pCi}]^{-1}$).

There are two classes of potential carcinogens identified at WAG 4 release sites: chemical carcinogens and radionuclides. These two classes of carcinogens are discussed separately in the following subsections.

6.4.1.1 Toxicity Assessment for Chemical Carcinogens. Evidence of chemical carcinogenicity originates primarily from two sources: (1) lifetime studies with laboratory animals and (2) human (epidemiological) studies. For most chemical carcinogens, animal data from laboratory experiments represent the primary basis for the extrapolation. Major assumptions arise from the necessity of extrapolating experimental results: across species (i.e., from laboratory animals to humans); from high-dose regions (i.e., levels to which laboratory animals are exposed) to low-dose regions (i.e., levels to which humans are likely to be exposed in the environment); and across routes of administration (i.e., inhalation versus ingestion). Federal regulatory agencies have traditionally estimated human cancer risks associated with exposure to chemical carcinogens on the administered-dose basis according to the following approach:

- The relationship between the administered dose and the incidence of cancer in animals is based on experimental animal bioassay results.
- The relationship between the administered dose and the incidence of cancer in the low-dose range is based on mathematical models.
- The dose-response relationship is assumed to be the same for both humans and animals, if the administered dose is measured in the proper units.

Effects from exposure to high (i.e., administered) doses are based on experimental animal bioassay results, while effects associated with exposure to low doses of a chemical are generally estimated from mathematical models.

For chemical carcinogens, EPA assumes a small number of molecular events can evoke changes in a single cell that can lead to uncontrolled cellular proliferation and tumor induction. This mechanism for carcinogenesis is referred to as stochastic, which means that there is theoretically no level of exposure to a given chemical that does not pose a small, but finite, probability of generating a carcinogenic response.

Because risk at low exposure levels cannot be measured directly either in laboratory animals or human epidemiology studies, various mathematical models have been proposed to extrapolate from high to low doses (i.e., to estimate the dose-response relationship at low doses). The three most frequently used models are (1) the one-hit model, (2) the log-probit model, and (3) the multistage model. The

one-hit model is based on the premise that a single molecule of a chemical can be the single event that precipitates tumor induction (Cornfield 1977). In other words, there is some finite response associated with any exposure. The log-probit model assumes that a response is normally distributed with the logarithm of the dose (Mantel et al. 1971). This theory seems to have little scientific basis, although some physiological parameters are lognormally distributed. This model usually yields much lower potency estimates because of the implied threshold at lower doses.

Currently, regulatory decisions are based on the output of the linearized multistage model (EPA 1989a). The basis of the linearized multistage model is that multiple events (versus the single-event paradigm of the one-hit model) may be needed to yield tumor induction. The linearized multistage model reflects the biological variability in tumor frequencies observed in animals or human studies (Crump et al. 1977). The dose-response relationship predicted by this model at low doses is essentially linear. Use of this model provides dose-response estimates intermediate between the one-hit and the log-probit models. It should be noted that the SFs calculated for nonradiological carcinogens using the multistage model represent the 95th percentile UCL on the probability of a carcinogenic response. Consequently, risk estimates based on these SFs are conservative estimates representing upper-bound estimates of risk where there is only a 5% probability that the actual risk is greater than the estimated risk.

Most models produce quantitatively similar results in the range of observable data, but yield estimates that can vary by three or four orders of magnitude at lower doses. Animal bioassay data are simply not adequate to determine whether any of the competing models are better than the others. Moreover, there is no evidence to indicate that the precision of low-dose risk estimates increases through the use of more sophisticated models. Thus, if a carcinogenic response occurs at the exposure level studied, it is assumed that a similar response will occur at all lower doses, unless evidence to the contrary exists.

Uncertainties in the toxicity assessment for chemical carcinogens are dealt with by classifying each chemical into one of several groups, according to the weight of evidence from epidemiological studies and animal studies, as follows (EPA, 1989a):

- Group A—Human carcinogen (sufficient evidence of carcinogenicity in humans)
- Group B—Probable human carcinogen (B1–limited evidence of carcinogenicity in humans; B2–sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)
- Group C—Possible human carcinogen (limited evidence of carcinogenicity in the animals and inadequate or lack of human data)
- Group D—Not classifiable as to human carcinogenicity (inadequate or no evidence)
- Group E—Evidence of noncarcinogenicity for humans (no evidence of carcinogenicity in adequate studies).

Table D-42 provides the SFs, in $(mg/kg/day)^{-1}$, and the weight-of-evidence for each WAG 4 COPC.

To obtain an estimate of total carcinogenic risk resulting from modeled exposures to carcinogens at the site, cancer risks are summed across all exposure routes for all carcinogens. Cancer risks from

exposure to multiple carcinogens across multiple pathways are assumed to be additive, based on EPA carcinogen risk assessment guidelines (EPA 1986).

6.4.1.2 Toxicity Assessment for Radionuclides. An extensive body of literature exists that describes the health effects of radionuclides on humans and animals. Intensive research by national and international commissions has resulted in the establishment of widely accepted limits to which workers and the public may be exposed without clinically detectable effects. This literature has resulted in EPA classifying all radionuclides as Group A carcinogens because radionuclides emit ionizing radiation, which, at high doses, has been associated with increased cancer incidence in humans. Human epidemiological data collected from the survivors of the Hiroshima and Nagasaki bomb attacks form the basis for the most recent extrapolation put forth by the National Academy of Science (NAS 1980). Conversely, for most nonradiological carcinogens, animal data from laboratory studies represent the primary basis for the extrapolation.

Another fundamental difference between the assessment of potential toxicity associated with exposure to radionuclide and nonradionuclide carcinogens is that SFs for radionuclides are typically best estimates (mean or median values rather than upper 95th-percentile values). Furthermore, in the past, risk factors for radionuclides have generally been based on fatalities (i.e., the number of people who actually died from cancer), while SFs for nonradiological carcinogens are based on incidence (i.e., the number of people who developed cancer). Finally, the SFs for radionuclides are expressed in different units, i.e., risk per $(pCi)^{-1}$ rather than $(mg/kg/day)^{-1}$.

Table D-42 lists SFs for all radionuclides identified at WAG 4 release sites. These nonthreshold SFs account for the following: the amount of radionuclide transported into the bloodstream, the decay of radioactive progeny within the body, the distribution and retention of the radionuclide and its progeny (if any) in the body, the radiation dose delivered to specific organs and tissues, and the age and sex of the exposed individuals (EPA 1995).

6.4.2 Toxicity Assessment for Noncarcinogenic Effects

Potential noncarcinogenic effects are evaluated by comparing daily intakes with chronic RfDs developed by the EPA. This section provides a definition of an RfD and discusses how it is applied in the OU 4-13 BRA. Table D-42 provides the RfD values for each of the COPCs identified at WAG 4 release sites.

A chronic RfD is an estimate of the daily exposure that can be incurred during a lifetime, without an appreciable risk of a noncancer effect being incurred in human populations, including sensitive subgroups (EPA 1989a). The RfD is based on the assumption that thresholds exist for noncarcinogenic toxic effects (e.g., liver or kidney damage). It is a benchmark dose operationally derived by the application of one or more order-of-magnitude uncertainty factors to doses thought to represent a lowest or no-observed-adverse-effect level (NOAEL) in humans. Thus, there should be no adverse effects associated with chronic daily intakes below the RfD value. Conversely, if chronic daily intakes exceed this threshold level, there is a potential that some adverse noncarcinogenic health effects might be observed in exposed individuals.

RfDs or SFs have not been developed by the EPA for the dermal exposure route. In the absence of these factors, the common practice has been to use the available toxicity measures for the oral route of exposure. This approach has been adopted in the BRA.

In evaluating the dermal pathway, the EPA recommends expressing chemical intake as absorbed dose and adjusting the oral toxicity measures to reflect absorbed dose (EPA 1989a). In deriving such

values, consistency is required between the type of dose that forms the basis of the oral toxicity and the type of dose that will be calculated by the dermal exposure models. Specifically, a distinction must be made between an administered dose or intake (i.e., the amount of chemical taken into the body) and the absorbed dose (i.e., the amount of chemical that crosses the body membranes and enters the bloodstream). Most of the toxicity measures available from the EPA are expressed as administered dose (i.e., intake) rather than dose at the tissue level (i.e., absorbed dose). The adjustment of the oral toxicity measure can be accomplished only if sufficient data are available in the principal laboratory studies or on the oral absorption efficiency in the species on which the toxicity measures are based. EPA notes that exposure estimates for absorption efficiency should not be adjusted if the toxicity values are based on administered doses (EPA 1989a).

For risk characterization purposes, the potential health effects of chronic exposure to noncarcinogenic compounds are assessed by calculating an HQ for each COPC. An HQ will be derived by dividing the estimated daily intake by a chemical-specific RfD as shown in the following equation:

$$HQ = Intake / RfD \quad (6\text{-}17)$$

where

HQ = Hazard quotient (unitless)

Intake = Chemical intake (mg/kg-day)

RfD = Reference dose (mg/kg-day).

An HQ greater than 1.0 indicates that exposure to a given chemical (at the concentrations and for the duration and frequencies of exposure estimated in the exposure assessment) may cause adverse health effects in exposed populations. However, the level of concern associated with exposure to noncarcinogenic compounds does not increase linearly as HQ values exceed 1.0. In other words, HQ values do not represent a probability or a percentage. For example, an HQ of 10 does not indicate that adverse health effects are 10 times more likely to occur than an HQ value of 1.0. All one can conclude is that HQ values greater than 1.0 indicate that noncarcinogenic health impacts are possible and that the higher the HQ value, the greater the concern about potential adverse health effects.

Consistent with RAGS, chemical-specific HQs are summed across exposure routes to calculate a HI for each COPC. Individual pathway HI values are then summed to determine a cumulative HI value for all exposure pathways and COPCs at each release site. This approach may result in a situation where a total HI value for a given release site may exceed 1.0 even though none of the chemical-specific HQ values at the release site exceed 1.0.

6.4.3 Toxicity Profiles

The following subsections present general and chemical-specific information on health effects relating to the COPCs evaluated in the BRA. All information presented in these subsections is from IRIS (EPA 1997b) unless otherwise specified. Chemical-specific toxicity values for each COPC discussed in these subsections are presented in Table D-4.

6.4.3.1 Arsenic. (CAS No. 7440-38-2; As; Mol. wt. = 75 g/mol; WoE = A; Oral SF = 1.5 mg/kg-day, oral RfD = 0.00003 mg/kg-day, inhalation SF = 50 mg/kg/day). Arsenic is a known carcinogen in humans. Ingestion is associated with increased incidence of skin cancer; lung cancer results from

inhalation. Insufficient data exist to determine carcinogenic effects in animals. Chronic exposure, either by ingestion or inhalation, is marked by malaise and fatigue. Changes in the skin include hyperkeratosis. Anemia, neuropathy, liver injury, and "blackfoot disease" can also result from chronic exposure.

Acute exposure to arsenic causes severe throat irritation, gastrointestinal disturbance, and muscle spasms. This is followed by vertigo, delirium, and coma. Facial edema may also be evident. Sensory loss and hematopoietic symptoms associated with acute exposure are usually reversible. The oral NOAEL and LOAEL are 0.0008 mg/kg-day and 0.014 mg/kg-day, respectively with critical effects of hyperpigmentation, keratosis, and possible vascular complications. The uncertainty factor and modifying factor for the NOAEL are 3 and 1, respectively (EPA 1997b).

6.4.3.2 Benzene. (CAS No. 71-43-2; C_6H_6; Mol. wt. = 78 g/mol; WoE = A; Oral RfD = 0.003 mg/kg-day; oral SF = 0.029 mg/kg-day^{-1}; inhalation RfD = 0.00171 mg/kg-day; and inhalation SF = 0.029 mg/kg-day^{-1}). Benzene is an aromatic hydrocarbon which occurs naturally in the environment and as a result of human activity. Benzene is utilized mainly in the manufacture of ethylbenzene (intermediate in synthesis of styrene for plastics), cumene (for the manufacture of phenol and acetone), and cyclohexane (for nylon resins). Environmental emissions of benzene, which are mainly airborne, arise from gasoline vapors, auto exhaust, and industrial production and applications. The highest exposure concentrations of benzene are found in industries utilizing benzene and benzene-containing products (ADEQ 1993).

Benzene is classified as having carcinogenic and noncarcinogenic effects in humans (EPA 1997b). Toxic effects in humans from inhalation and ingestion exposures to benzene have resulted in death from respiratory arrest, CNS depression, and cardiac collapse. Inhalation exposures to humans have also resulted in hematological (deficit in the circulating blood cells, aplastic anemia, leukemia), immunological (changes in the blood levels of antibodies and circulating leukocytes), neurological (dizziness, tremor, delirium, unconsciousness), developmental (chromatid breaks, sister chromatid exchange in children of exposed females), and reproductive (impaired fertility, menstrual disorder, spontaneous abortion) effects, particularly in studies of occupationally-exposed groups. With human ingestion exposures, GI (gastritis, pyloric stenosis), hematological (decrease in erythrocytes and leukocytes), dermal (swelling and edema of skin), and neurological (vertigo, muscular incoordination, unconsciousness) effects have also been reported. Dermal exposures have resulted in skin irritation (ADEQ 1993). The inhalation NOAEL for benzene is 2.35 mg/kg-day with a critical effect of hematological impairment. The uncertainty factor for the NOAEL is 100 (EPA 1997b).

In addition, hepatic (alteration of hepatic drug metabolism), immunological (decrease in peripheral blood leukocytes), and developmental (reduction in the weight of rodent pups) effects have also been noted in animals with ingestion exposure (ADEQ 1993).

Epidemiological studies have shown an association between inhalation exposure to benzene and the development of leukemia (particularly the acute myeloid form) and lymphopoietic cancer in humans. Animal studies have supported the finding of leukemia with inhalation exposure and have also shown lymphomas with ingestion. Skin tumors have been demonstrated with dermal exposures. Genotoxic effects (chromosomal aberrations) in occupational groups have also been documented with inhalation and dermal exposures (ADEQ 1993).

6.4.3.3 Benzo(a)anthracene. (CAS No. 56-55-3; $C_{18}H_{12}$; Mol. wt = 228.3 g/mol; WoE = B2; Oral SF = 0.73 mg/kg-day^{-1}; Inhalation SF = 0.31 mg/kg-day^{-1}; Dermal SF = 0.73 mg/kg-day^{-1}). Benzo(a)anthracene is a member of a class of chemical compounds known as Polycyclic Aromatic Hydrocarbons (PAHs). PAHs are a group of chemicals formed during the incomplete burning of coal, oil and gas, garbage, and other organic substances. PAHs are used for research purposes, in medicines, and

to make dyes, plastics, and pesticides. They are found throughout the environment in air, water, and soil. PAHs can occur as a result of anthropogenic or natural activities (e.g., forest fires)(ATSDR 1990a).

PAHs tend to sorb strongly to soil and organic matter including other PAHs. Higher molecular weight PAHs tend to have lower solubilities in water. Hydrophobic PAHs have a high affinity for binding to organic matter and have relatively high biotransformation rates. The dominant mechanism of PAH removal from soil is microbial degradation. PAHs can persist in soils for years (ATSDR 1990a).

PAHs have background levels in air between 0.02 and 1.2 mg/m^3 in rural areas and between 0.15 and 19.3 mg/m^3 in urban areas. The background level of PAHs in drinking water ranges from 4 to 24 ng/L. PAHs are present in tobacco smoke, smoke from wood and creosote-treated wood products, cereals, grains, flour, bread, vegetables, fruits, meats, processed or pickled food, and beverages. The average U.S. diet contains less than 2 ppb of total PAHs (ATSDR 1990a).

Benzo(a)anthracene is a probable human carcinogen, based on sufficient data from animal bioassays in which benzo(a)anthracene produced tumors in mice exposed by gavage, intraperitoneal, subcutaneous or intramuscular injection, in addition to topical application. Although there are no human data that specifically link its exposure to human cancers, benzo(a)anthracene is a component of mixtures that have been associated with human cancer such as coal tar, soots, coke oven emissions and cigarette smoke (EPA 1997). Exposure to benzo(a)anthracene through these components may be associated with cancer of the liver, mammary gland and respiratory and gastrointestinal tracts. The oral NOAEL is 150 mg/kg-day based on a four-day rat study. Critical effects of the study involved gastrointestinal, hepatic and renal toxicity (ATSDR 1990a).

6.4.3.4 Benzo(b)fluoranthene. CAS No. 205-99-2; $C_{20}H_{12}$; Mol. Wt = 252.3 g/mol; WoE = B2; Oral SF = 0.73 $mg/kg\text{-}day^{-1}$; Inhalation SF = 0.31 $mg/kg\text{-}day^{-1}$; Dermal SF = 0.73 $mg/kg\text{-}day^{-1}$). Benzo(b)fluoranthene, a member of the PAH class of chemical compounds, is a probable human carcinogen, based on sufficient data from animal bioassays in which benzo(b)fluoranthene produced tumors in mice after lung implantation, intraperitoneal or subcutaneous injection, in addition to topical application. Although there are no human data that specifically link its exposure to human cancers, benzo(b)fluoranthene is a component of mixtures that have been associated with human cancer such as coal tar, soots, coke oven emissions and cigarette smoke (EPA 1997). The oral NOAEL is 150 mg/kg-day based on a four-day rat study. Critical effects of the study involved gastrointestinal, hepatic and renal toxicity (ATSDR 1990a). See Section 6.4.3.1 for a general discussion of the chemical properties and toxicity of the PAH class of compounds.

6.4.3.5 Benzo(g,h,i)perylene. (CAS No. 191-24-2; $C_{22}H_{12}$; Mol. Wt = 276 g/mol; WoE = D; no toxicity values available). Benzo(g,h,i)perylene, a member of the PAH class of compounds, is classified as a noncarcinogen. It is not produced commercially in the United States and has no known use (ATSDR 1990a). Benzo(g,h,i)perylene's most common route of exposure is through inhalation, but oral exposure to contaminated drinking water, food, and soil is also possible. Because of its high molecular weight, benzo(g,h,i)perylene is not as mobile as other PAHs such as phenanthrene. The oral NOAEL is 150 mg/kg-day based on a four-day rat study. Critical effects of the study involved gastrointestinal, hepatic and renal toxicity (ATSDR 1990a). See Section 6.4.3.3 for a general discussion of the chemical properties and toxicity of the PAH class of compounds.

6.4.3.6 Chlorodifluoromethane. (CAS No. 75-45-6; $CHClF_2$; Mol. wt. = 86.47 g/mol; WoE is not available; Inhalation RfD = 14.3 mg/kg-day). Chlorodifluoromethane is a colorless, nonflammable gas with a slight etheral odor and is also known as Halocarbon 22. It is used by the semiconductor industry for plasma etching.

Chlorodifluoromethane is classified as having noncarcinogenic effects in humans from chronic exposures (EPA 1997b). Potential health effects from acute exposure to chlorodifluoromethane are through inhalation, dermal contact, and ingestion. Inhalation causes possible dizziness, drowsiness, and throat irritation at levels above 1,000 ppm. Minimal effects were observed below 1,000 ppm. Unconsciousness and death can occur at levels above 10,000 ppm. Inhalation of chlorodifluoromethane has resulted in cellular necrosis (EPA 1997b). As liquid or vapor, chlorodifluoromethane can cause eye irritation and prolonged or repeated skin contact can cause freezing of the tissue. Single dose ingestion is low to moderate, but can be aspirated into the lungs causing chemical pneumonia if vomited. The inhalation LC_{50} is 26,200 ppm/4 hours (Tech Spray 1997). The inhalation NOAEL and LOAEL are 35,370 mg/m^3 and 176,800 mg/m^3, respectively with critical effects of increased kidney, adrenal, and pituitary weights. The uncertainty factor and modifying factor for the NOAEL are 100 and 1, respectively (EPA 1997b).

6.4.3.7 Di-n-butylphthalate. (CAS No. 84-74-2; $C_{16}H_{22}O_4$; Mol. wt. = 278.35 g/mol; WoE = D; Oral RfD = 0.1 mg/kg-day). Di-n-butylphthalate is primarily used as a plasticizer for epoxy resins and polyvinyl chloride (PVC). Other applications include use as an adjusting agent for lead chromate pigments; use as a concrete additive; use in polyvinyl acetate emulsions, use as an insect repellent; and use in cosmetics (DTIC 1990a). The primary exposure pathway of concern from a soil-water systems is the migration to groundwater drinking water supplies (although this compound is strongly sorbed to soil and such migration has not been observed in the past). Inhalation resulting from volatilization from surface soils is not expected to be significant (DTIC 1990a).

Di-n-butylphthalate is classified as having noncarcinogenic effects in human from chronic exposures (EPA 1997b) and does not exhibit carcinogenic effects in humans. Acute exposure via ingestion of di-n-butylphthalate may cause nausea, dizziness, light sensitivity, and watering and redness of the eyes. Heated vapors may irritate the eyes, nose, and throat. Accidental ingestion caused delayed effects by several hours which include: nausea, vomiting and dizziness, followed by headache, pain and eye irritation, lacrimation, photophobia and conjuctivitis. Recovery was complete within 2 weeks. Eye contact has caused immediate, severe, stinging pain, but no appreciable damage. Heated vapors may be eye, nose and throat irritant, but relatively non-irritating to the skin (DTIC 1990a).

The reproductive effects of chronic exposure to di-n-butylphthalate have not been verified; pregnancy/neonate effects include teratogen/testicular atrophy, and genotoxicity effects are negative based on limited evidence (DTIC 1990a). The oral NOAEL and LOAEL are 125 mg/kg-day and 600 mg/kg bw/day, respectively with a critical effect of increased mortality. The uncertainty factor and modifying factor for the NOAEL are 1000 mg/kg-day and 1, respectively (EPA 1997b)

6.4.3.8 Ethylbenzene. (CAS No. 100-41-4; C_8H_{10}; Mol. wt. = 106.17 g/mol; WoE = D; Oral RfD = 0.1 mg/kg-day; and inhalation RfD = 0.286 mg/kg-day). Ethylbenzene is an aromatic hydrocarbon which occurs naturally in the environment and as a result of human activity. Although ethylbenzene is used mainly as a solvent, other uses include styrene production, as well as use in asphalt, naptha, and fuels. Environmental emissions of ethylbenzene, which are mainly airborne, arise from industrial processes or in the combustion of fossil fuels. The main route of human exposure is related to chronic inhalation of low-level ethylbenzene concentrations due to direct release of ethylbenzene into the air by burning fossil fuels or using paints, inks, and insecticides containing ethylbenzene. Ingestion of ethylbenzene is also a potential route of exposure due to trace amounts found in many open water supplies.

Ethylbenzene is classified as having noncarcinogenic effects to human (EPA 1997b) and does not exhibit carcinogenic effects in humans. Acute exposure to ethylbenzene results in neurological and respiratory depressions, in addition to eye and throat irritation. Several studies suggest that target organs

may include the liver, kidney, and hematopoietic system, although results are inconclusive (ATSDR 1990b). Systemic, immunological, neurological, reproductive, developmental, and genotoxic effect that may result from inhalation exposure to ethylbenzene are summarized below.

Death in humans resulting from chronic, low-level exposure to ethylbenzene is unlikely (ATSDR 1990b). Ethylbenzene exhibits several systemic effects in humans. Moderate upper respiratory irritation accompanied by chest constriction has been reported in several human inhalation cases. Severe respiratory effects in humans could result following inhalation exposure to high doses of ethylbenzene. Ethylbenzene-induced hematological effects following inhalation exposure have been observed in lab animals, but these effects are unknown in humans. No hepatotoxic effects in humans have been reported. Hepatic effects in mice and rats exposed orally and by inhalation may affect humans, but studies have been inconclusive thus far. Possible renal effects (enzyme changes, organ weight increase and tubular swelling) could occur in humans exposed to high doses of ethylbenzene, but these studies are inconclusive (although animal evidence exists) (ATSDR 1990b).

Neurological effects from chronic inhalation exposure to ethylbenzene in humans is unknown. Acute inhalation at high concentrations principally affects the CNS in humans. These effects include dizziness and vertigo. Complete CNS recovery is possible following acute exposure (ATSDR 1990b).

No developmental effects has been indicated following human exposure to ethylbenzene. Animal (rat) studies indicate fetotoxic effects exist at doses which also induce toxic effects on the dams (ATSDR 1990b). No human studies are available regarding reproductive effects. Animal studies report reproductive effects, but data results were insufficient (ATSDR 1990b). Genotoxic effects of ethylbenzene in humans are not known, although two studies do suggest ethylbenzene causes an increase in the potential for genotoxicity in humans (ATSDR 1990b). The oral NOAEL and LOAEL are 97.1 mg/kg-day and 291 mg/kg-day, respectively with critical effects of liver and kidney toxicity. The uncertainty factor and modifying factor for the NOAEL are 1000 and 1, respectively (EPA 1997b). The inhalation NOAEL and LOAEL are 434 mg/m^3 and 4340 mg/m^3, respectively with a critical effect of developmental toxicity. The uncertainty factor and modifying factor for the NOAEL are 300 and 1, respectively (EPA 1997b).

6.4.3.9 Lead. (CAS No. 7439-92-1; Pb; Mol. wt. = 207 g/mol; WoE = B2; no toxicity values available). Lead is a naturally occurring bluish-gray metal. It is used in the production of batteries, ammunition, various metal products (such as sheet lead, solder, brass and bronze products, and pipes) and in ceramic glazes and paints. Tetraethyl lead and tetramethyl lead were historically used as a gasoline additive, but these uses were discontinued in 1996 (ATSDR 1997).

Lead is classified as a probable human carcinogen based on sufficient evidence in animals, but inadequate evidence in humans (EPA 1997b). Acute exposure to lead can occur primarily through ingestion, inhalation and dermal contact. Exposure may cause CNS depression, weakness in extremities, and increased blood pressure. Exposure to elevated concentrations may cause liver and neurological toxicity (ATSDR 1997).

Lead produces neurotoxic and behavioral effects particularly in children. However, the EPA believes that it is inappropriate to set an RfD for lead and its inorganic compounds because the agency believes that some of the effects may occur at such low concentrations as to suggest no threshold. The EPA has also determined that lead is a probable human carcinogen (classified at B2) (ASTM 1995).

6.4.3.10 Mercury. (CAS No. 7439-97-6; Hg; Mol. wt. = 201 g/mol; WoE = D; Oral RfD = 0.0003 mg/kg-day, inhalation RfD = 0.0000857 mg/kg-day). The chemistry of mercury in the environment is complex, not only because of its various oxidation states but also because of biotic and

abiotic methylation and demethylation processes, complexation with organic and inorganic ligands, and the differential solubility and volatility of various forms. Speciation is a major determinant of the fate, bioavailability, absorption, and toxicologic characteristics of mercury compounds.

Although the generally more toxic organic forms of mercury are unlikely to persist in the environment, they (in particular, methylmercury) may be formed in biotic tissues and are known to biomagnify through ecosystems, particularly aquatic systems (Wren 1986; Scheuhammer 1987).

Because of its chemical stability and lipophilicity, methylmercury readily penetrates the blood-brain barrier. The central nervous system is thus a major target organ in both mammals and birds. However, reproductive effects have been reported at even lower doses. Methylmercury can be converted to inorganic mercury both in tissues and by microflora in the gut. The homolytic cleavage of the mercury-carbon bond leads to generation of reactive intermediates, e.g., methyl and metal radicals, which cause cellular damage (Wren 1986; Scheuhammer 1987; Manzo et al. 1992). The inhalation NOAEL and LOAEL are none and 0.009 mg/m^3, respectively with critical effects of hand tremor, increases in memory disturbances, and slight subjective and objective evidence of autonomic dysfunction. The uncertainty factor and modifying factor for the NOAEL are 30 and 1, respectively (EPA 1997b).

6.4.3.11 Phenanthrene. (CAS No. 85-01-8; $C_{14}H_{10}$; Mol. wt. = 178.08 g/mol; WoE =D; no toxicity values available). Phenanthrene, a member of the PAH class of chemical compounds, is noncarcinogenic, producing negative cancer results when tested. Phenanthrene is virtually insoluble in water, but is degraded by microbes in the soil. Phenanthrene is not produced commercially in the United States and there is no known use for phenanthrene except as a research chemical. The oral NOAEL is 150 mg/kg-day with critical effects of the gastrointestinal, hepatic, and renal systems. The NOAEL was done for a rat for four days (ATSDR 1990a). See Section 6.4.3.1 for a general discussion of the chemical properties and toxicity of the PAH class of compounds.

6.4.3.12 Phenol. (CAS No. 108-95-2; C_6H_6O; Mol. wt. = 94.11 g/mol; WoE = D; Oral RfD = 0.6 mg/kg-day). Phenol is primarily used as a chemical intermediate in the synthesis of organic chemicals (primarily phenolic resins). Less significant uses of phenol involve use as a solvent in petroleum refining and as a disinfectant (DTIC 1990a). The primary pathway of concern from soil-water systems is the migration of phenol to groundwater supplies of drinking water. Data suggests that such migration has occurred in the past. The consumption of fish or other organisms is not expected to be a significant route of exposure (DTIC 1990a).

Phenol is classified as having noncarcinogenic effects to humans from chronic exposures and does not exhibit carcinogenic effects in humans (EPA 1997b). Chronic effects include liver and kidney damage, in addition to skin discoloration. Pregnancy/neonate effects are teratogenic only at maternally lethal doses; studies show fetotoxic effects occur at doses that are not toxic to the female animal during pregnancy, and genotoxic results are unclear due to conflicting evidence (DTIC 1990a). Phenol is readily absorbed from all routes of entry, distributed throughout the body, metabolized and rapidly excreted. The most frequent adverse effects from phenol reported in humans resulted from skin contact. Signs and symptoms can develop rapidly with serious consequences including shock, convulsions, cyanosis, coma and death. Direct contact with the skin results in chemical burns (DTIC 1990a).

Acute exposure to phenol has a marked corrosive action on tissue. On contact with the eyes, it may cause severe damage and blindness. On skin, it induces anesthesia and blanching of the exposed area. If not removed promptly, it may cause a severe burn and systemic intoxication. Systemic effects, which can result from any route of exposure, include paleness, weakness, sweating, headache, ringing of the ears, cyanosis, shock, excitement, frothing of the nose and mouth, and death (DTIC 1990a). The oral NOAEL

and LOAEL are 60 mg/kg-day and 120 mg/kg-day, respectively with a critical effect of reduced fetal body weight in rats.

6.4.3.13 Tetrachloroethene. (CAS No. 127-18-4; C_2Cl_4; Mol. wt. = 165.85 g/mol; WoE = C-B2; Oral RfD = 0.01 mg/kg-day; oral SF = 0.052 mg/kg-day^{-1}; inhalation SF = 0.00203 mg/kg-day^{-1}). Tetrachloroethene (PCE) is used primarily in the dry cleaning industry. It is also used in cold cleaning, vapor degreasing of metals, and as a chemical intermediate in the synthesis of fluorocarbons. Minor applications include various manufacturing and industrial processes as well as medicinal uses (DTIC 1990a).

The primary exposure pathway of concern from a soil-water system is the migration of PCE to groundwater used as sources for drinking water. Inhalation resulting from volatilization from surface soils and drinking water may also be important (DTIC 1990a).

PCE is classified as a possible human carcinogen. Chronic effects include liver and kidney toxicity, and both pregnancy/neonate effects and genotoxicity effects are negative (DTIC 1990a). Acute exposure via ingestion and inhalation can cause nausea, vomiting, headache, dizziness, drowsiness, and tremors. Skin contact with liquid causes irritation and blistering. Both the liquid and vapor are eye irritants (DTIC 1990a). The oral NOAEL and LOAEL are 14 mg/kg-day and 100 mg/kg-day, respectively with critical effects of hepatotoxicity in mice and weight gains in rats. The uncertainty factor and modifying factor for the NOAEL are 1000 and 1, respectively (EPA 1997b).

6.4.3.14 Toluene. (CAS No. 108-88-3; $C_6H_5CH_3$; Mol. wt. = 92.15 g/mol; WoE = D; Oral RfD = 0.2 mg/kg-day; and inhalation RfD = 0.114 mg/kg-day). Toluene is an aromatic hydrocarbon which occurs naturally in the environment and as a result of human activity. Toluene is used as a solvent in paint and paint thinners, in addition to various printing and leather tanning processes. Environmental air emissions of toluene arise from automobile exhaust, in addition to petroleum, iron-coke, and styrene production. Toluene can enter soil, surface water, and ground water from spills and leaking underground storage tanks. The main route of human exposure is related to inhalation of low-level toluene concentrations due to the direct release of toluene into the air, soil, surface water, and ground water.

Toluene is classified as having noncarcinogenic effects in humans (EPA 1997b) and does not exhibit carcinogenic effects in humans. Acute inhalation exposure to toluene results primarily in respiratory tract irritation. High concentrations of toluene under chronic exposure in rats indicated respiratory irritation and pulmonary lesions (ATSDR 1994). Systemic, immunological, neurological, reproductive, developmental, and genotoxic effect that may result from inhalation exposure to toluene are summarized below.

Acute inhalation exposure at elevated concentrations resulted in heart rhythm alterations when tested on animals. Chronic exposure studies in humans indicated possible gastrointestinal irritation. Hematological effects are not expected to occur in humans due to the lack of response in several animal studies. Hepatic effects have not been observed in humans, but there is a possibility that the liver's ability to metabolize xenobiotics similar to toluene is affected. Renal effects have not been observed in workers exposed to low level, chronic exposure to toluene (Askergren *et al.*, 1981; Neilsen *et al.*, 1985). Eye and skin irritations have been reported after human exposure to toluene (ATSDR 1994).

Animal studies indicate immunological effects such as decreased thymus weight, lymphocyte culture responses and antibody plaque-forming cell responses, but no human data is currently available (ATSDR 1994). The primary neurological effect of inhalation exposure in humans is CNS depression with the following symptoms: fatigue, confusion, and incoordination, as well as impairment in reaction time, perception, and motor control. High concentration, occupational exposure has led to residual or

permanent CNS effects. Toluene is ototoxic in both animals and humans. Hearing deficits were identified when workers were chronically exposed to 100 ppm of toluene (ATSDR 1994). It is difficult to speculate whether reproductive effects occur in women exposed to toluene (ATSDR 1994).

Human developmental effects are inconclusive, but the occurrence of neurobehavioral effects and possible fetotoxicity in animals is a cause of concern to humans (ATSDR 1994). Genetic assays generally indicate that toluene is nonmutagenic and nongenotoxic (ATSDR 1994). The oral NOAEL and LOAEL are 223 mg/kg-day and 446 mg/kg-day, respectively with critical effects of changes in liver and kidney weights. The uncertainty factor and modifying factor for the NOAEL are 1000 and 1, respectively (EPA 1997b). The inhalation NOAEL and LOAEL are none and 332 mg/m^3, respectively with a critical effect of neurological effects. The uncertainty factor and modifying factor for the NOAEL are 300 and 1, respectively (EPA 1997b).

6.4.3.15 TPH-Diesel. (CAS No. none; TPH is a mixture; Mol. wt. = none; WoE not available; Oral RfD = 0.6 mg/kg-day). Diesel is a blue liquid mixture used mainly as a fuel in machinery and automobiles. Diesel is incompatible with oxygen and strong oxidizing agents, and will ignite in the presence of heat, sparks, or flame. Acute effects from overexposure are mild eye irritation, severe skin irritation, respiratory and gastrointestinal irritation. When diesel burns, the exhaust when inhaled causes short term carboxyhemoglobinemia and long term lung cancer in humans (Phillips 66 1997). The oral LD_{50} in rats is 9 mL/kg and dermal LD_{50} in rabbits is >5 mL/g (Phillips 66 1997).

Total Petroleum Hydrocarbons as diesel (TPH-diesel) is comprised of hydrocarbons in the C_{10} to C_{20} range. Because of their higher molecular weights, constituents in these products are less volatile, less water soluble, and less mobile than gasoline-range hydrocarbons. About 25 to 35% of No. 2 fuel oil is composed of aromatic hydrocarbons, primarily alkylated benzenes and naphthalenes. The BTEX concentrations are generally low (ASTM 1995).

6.4.3.16 1,1,1-Trichloroethane. (CAS No. 71-55-6; $C_2H_3Cl_3$; Mol. Wt = 133.42 g/mol; WoE not available; Oral RfD = 0.02 mg/kg-day; Inhalation RfD = 0.286 mg/kg-day). 1,1,1-Trichloroethane (1,1,1-TCA) is widely used as a cleaning solvent because of its nonflammability and solvency properties. As of 1985, approximately 28% of the total production was used in vapor degreasing and 41% in cold cleaning. Common solvent uses include cleaning of electrical equipment, motors, electronic components and instruments, missile hardware, photographic film, printed circuit boards, upholstery, and various metal and plastic parts during manufacture. It is also used as a solvent for adhesives and coatings, photoresist polymers, textile dyes, as coolant and lubricant in metal cutting oils ,as a component in inks and drain cleaners, and as a chemical intermediate in the production of vinylidene chloride. It has minor use in aerosols where it acts both as a vapor pressure depressant and as a solvent and carrier for the active ingredients (DTIC 1990b).

1,1,1-TCA is expected to be fairly mobile in the soil/groundwater system, particularly is soils of low organic carbon where adsorption is low. Volatilization is an important removal process for near-surface contamination (DTIC 1990b).

1,1,1-TCA is classified as having noncarcinogenic effects in humans from chronic exposure (EPA 1997b) and does not exhibit carcinogenic effects in humans. Chronic effects include liver toxicity. Pregnancy/neonate effects are negative and genotoxic effects are unclear due to conflicting results (DTIC 1990b). Acute exposure leads to dizziness, drowsiness, lack of coordination, increased reaction time and irregular heart beat. Both liquid and vapor are eye irritants. Skin contact may produce dermatitis. Depression of the central nervous system is the primary toxic effect in humans who have been subjected to short-term, high-level inhalation exposure. Inhalation of 450 ppm for 8 hr caused eye, nose, and throat irritation. Acute inhalation exposures can also adversely effect the cardiovascular system.

Numerous deaths have been attributed to deliberate or occupational inhalation to 1,1,1-TCA. In the majority of human fatalities, death results from CNS depression, edema and pulmonary congestion (DTIC 1990b). The single oral dose NOAEL is 1400 mg/kg-day with a critical effect of depressed hepatic metabolism. The uncertainty factor for the NOAEL is 100 (EPA 1997b).

6.4.3.17 Xylenes, mixed. (CAS No. 1330-20-7; C_8H_{10}; Mol. wt. = 106 g/mol; WoE = D; Oral RfD = 2 mg/kg-day). Xylene is an aromatic hydrocarbon which consists of three isomers (ortho, meta, and para). Xylene occurs naturally in the environment, in addition to a product of human activity. Xylene is a product of petroleum, coal, and forest fires. Xylene is used as a solvent in the printing, rubber, and leather industries. Xylene is also found in airplane fuel, gasoline, and cleaning agents. Xylene is used as a material in the paint, chemical, plastic, and synthetic fiber industries, in addition to an ingredient in the coating of fabrics and papers. Xylene emissions occur primarily from industrial sources, auto exhaust, and solvent use. Release from the use, storage, and transport of petroleum and xylene products also serves as possible routes for human exposure (ATSDR 1990c).

Xylene is classified as having noncarcinogenic effects in humans (EPA 1997b) and does not exhibit carcinogenic effects in humans. Acute exposure to high concentrations of xylene can cause irritation to the skin, eyes, nose, and throat; breathing difficulty; impaired pulmonary function; delayed response to visual stimulus; impaired memory; stomach discomfort; and possible hepatic and nephrotic effects. Both acute and chronic exposure to high concentrations of xylene can cause CNS effects such as dizziness, headaches and general confusion. Systemic, immunological, neurological, reproductive, developmental, and genotoxic effect that may result from inhalation exposure to xylene are summarized below (ATSDR 1990c).

Animal studies show observed hepatic, nephrotic, pulmonary, cardio, and CNS effects. Chronic, low-level concentrations of Xylene have not been studied in depth. Carcinogenic effects on animals have not been determined, although xylene is thought to be a possible human teratogen. Primary target organs: liver and CNS (ATSDR 1990c).

Xylene can be fatal to both humans and animals following inhalation and oral exposure. No fatal dermal exposure cases have been reported in humans. Death in humans and animals appears to be caused by either respiratory failure or ventricular fibrillation. The amount of xylene necessary to cause death is relatively large in both animals and humans, and reports of death in humans following inhalation occurred in areas of poor ventilation. Therefore, it is highly unlikely that inhalation or ingestion of the small amounts of xylene present in contaminated water or air would pose a fatal risk (ATSDR 1990c).

In humans, acute inhalation of xylene produced nose and throat irritation. Severe lung congestion with pulmonary hemorrhages and edema was noted in a worker who died following acute inhalation. In addition, chronic occupational exposure to xylene vapors was associated with labored breathing and impaired pulmonary function. Symptoms such as nausea, vomiting and gastric discomfort have been noted in workers following inhalation (ATSDR 1990c).

Available human studies suggest possible evidence that inhalation exposure to solvent mixtures containing xylene may increase the risk of developing renal dysfunction or renal damage resulting in increased blood urea concentrations, decreased urinary clearance of endogenous creatinine, increased lysozymuria, increased urinary levels of B-glucuronidase, and increased urinary excretion of albumin, erythrocytes, and leukocytes. No human data is available regarding the renal toxicity of xylene following oral or dermal exposure. Human dermal exposure causes irritation, dryness, scaling, and vasodilation of the skin.

Results of experimental studies with animals provide further evidence that mixed xylene and individual isomers are neurotoxicants following inhalation exposure. Neurotoxicity symptoms in animals include narcosis, prostration, incoordination, tremors, muscular spasms, labored breathing, behavioral changes, hyperactivity, elevated auditory thresholds, hearing loss, changes in brain enzyme activity and changes in brain protein levels (ATSDR 1990c).

Various assays indicate that mixed xylene and xylene isomers are nongenotoxic. No data were available regarding the development of cancer in humans following inhalation, oral, or dermal exposure to mixed xylene or xylene isomers (ATSDR 1990c). The oral NOAEL is 179 mg/kg-day with critical effects of hyperactivity, decreased body weight, and increased mortality (males). The uncertainty factor and modifying factor for the NOAEL are 100 and 1, respectively (EPA 1997b).

6.4.3.18 Radionuclides. The EPA classifies all radionuclides as "Group A" carcinogens (i.e., WoE =A) because radionuclides emit ionizing radiation and because of the extensive weight-of-evidence provided by epidemiological studies of radiation-induced cancers in humans. Ionizing radiation has sufficient energy to interact with matter and produce an ejected electron and a positively charged ion. In addition, ionizing radiation can produce new chemical species, known as free radicals, from water in the body. Free radicals are highly reactive and may combine with other elements or compounds within a cell to produce toxins or otherwise disrupt a cell's chemical balance. These disruptions may result in mutations or other deleterious effects.

Radionuclides are characterized by the type and energy level of the radiation emitted. Radionuclides contained in WAG 4 soils produce external radiation exposures principally through the production of beta, gamma, and alpha radiation.

The general health effects of radiation can be divided into stochastic and nonstochastic effects (i.e., those health effects not related to threshold dose and those related to threshold dose). Developing cancer from exposure to any amount of radiation is a stochastic effect. Examples of nonstochastic effects include acute radiation syndrome and cataract formation, both of which occur only at high levels of exposures.

Radiation can damage cells in different ways. First, radiation can cause damage to the strands of genetic material, DNA, in a cell. The cell may not be able to recover from this type of damage, or the cell may live on in a functionally abnormal condition. If the abnormally functioning cell divides and reproduces, a tumor or mutation in the tissue may develop. The rapidly dividing cells that line the intestines and the stomach and the cells that make blood in the bone marrow are very sensitive to this kind of damage. Organ damage results from the damage caused to the individual cells. This type of damage has been reported with doses of 10 to 500 rem. Acute radiation sickness is seen only after doses of greater than 50 rem. This dose is usually only received by personnel in proximity to serious nuclear accidents. Principal adverse effects associated with exposure to ionizing radiation are carcinogenicity, mutagenicity, and teratogenicity.

When cells damaged by radiation are reproductive cells, genetic damage can occur in the offspring of the person exposed. The developing fetus is especially sensitive to radiation. The type of malformation that may occur is related to the stage of fetal development and the cells that are differentiating at the time of exposure. Radiation damage to children exposed while in the womb is related to the dose that the pregnant mother received. Mental retardation is another possible effect of fetal radiation exposure.

In the following subsections f_1 is the fractional absorption of a stable radionuclide from the gastrointestinal tract and the class D, W, and Y describes the clearance time of inhaled radioactive

materials from the lung. The class D applies to a half-time of less than 10 days, class W applies to a half-time from 10 to 100 days, and class Y applies to a half-time of greater than 100 days. The following subsections provide additional information about the specific radionuclide COPCs at WAG 4.

6.4.3.18.1 Americium-241. (CAS No. 014596-10-2; Atomic No. 95; Mol. wt. = 241 g/mol; Half-life = 432 years; Ingestion SF = 3.28E-10 risk/pCi; inhalation SF = 3.85E-08 risk/pCi; and external SF = 4.59E-09 risk/pCi). Am-241 is produced by the beta decay of Pu-241. This isotope has been distributed widely in the environment as a result of nuclear weapons fallout. Am-241 decays by alpha emission, which makes the isotope important for internal exposure, whether it is ingested or inhaled. The alpha decay is accompanied by emission of gamma of radiation 60 keV with an abundance of 36%, which is of concern where Am-241 is concentrated, but is not important at environmental levels. The International Committee on Radiological Protection (ICRP) has assigned a value of 5.00E–04 to f_1, the fractional absorption of americium from the gastrointestinal tract, for all compounds of americium. For inhalation exposures, the ICRP recommends assigning all compounds of americium to inhalation Group W. Most (90%) of the americium entering the blood stream is deposited in the liver and the bone, with only a small amount being deposited in human reproductive organs. The biological half-lives in the liver and the bone are 40 and 100 years, respectively. The amount deposited in reproductive organs is considered to remain permanently.

6.4.3.18.2 Actinium-228. (CAS No. 014331-83-0; Atomic No. 89; Mol. wt. = 228 g/mol; Half-life = 6.13 hours; Ingestion SF = 1.62E-12 risk/pCi; inhalation SF = 3.27E-11 risk/pCi; and external SF = 3.28E-06 risk/pCi). Ac-228 is a beta-emitting member of the decay chain of naturally occurring Th-232. It has a half-life of 6.13 hours and exists in secular equilibrium with the long-lived parent radionuclides of the decay chain.

ICRP (1979) has assigned a value of 0.001 to f_1, the fractional absorption of actinium from the gastrointestinal tract. Oxides and hydroxides are assigned by ICRP to inhalation class Y, halides and nitrates are assigned class W, and other common compounds are assigned class D. Actinium primarily deposits in the mineral bone and the liver, with biological half-lives of 100 years and 40 years, respectively.

6.4.3.18.3 Barium-133. (CAS No. 013981-41-4; Atomic No. 56; Mol. wt. = 133 g/mol; Half-life = 10.5 years; Ingestion SF = 2.70E-12 risk/pCi; inhalation SF = 4.03E-12 risk/pCi; and external SF = 9.15E-07 risk/pCi). Ba-133 has a physical half-life of 10.53 years and decays by electron capture to cesium-133. ICRP has assigned a value of 0.1 to f_1, the fractional absorption of barium from the gastrointestinal tract. All common compounds are assigned inhalation class D by ICRP. Barium primarily deposits in the bone, and ICRP assumes that it is distributed throughout the volume of mineral bone.

6.4.3.18.4 Bismuth-212. (CAS No. 014913-49-6; Atomic No. 83; Mol. wt. = 212 g/mol; Half-life = 60.55 minutes; Ingestion SF = 6.20E-13 risk/pCi; inhalation SF = 3.65E-11 risk/pCi; and external SF = 6.67E-07 risk/pCi). Bi-212 is an alpha-and beta-emitting member of the decay chain associated with Th-228, which is part of the larger decay chain of naturally occurring Th-232. Bismuth-212 has a physical half-life of 60.5 minutes and exists in secular equilibrium with the long-lived parent radionuclides of the decay chain. ICRP has assigned a value of 0.05 to f_1, the fractional absorption of bismuth from the gastrointestinal tract. Bismuth nitrate is assigned by ICRP to inhalation class D, and all other compounds are assigned class W. Bismuth is primarily deposited in the kidneys, with different fractions assumed to clear with biological half-lives of 0.6 and 5 days.